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Abstract

Considerable evidence suggests a role of beta-band oscillations in voluntary movements. However, most of the studies linking beta power to motor performances are based on data averaged across trials that ignore the fast dynamics of oscillatory activity and variations in motor responses. Recently, emphasis has shifted from the functional implications of the mean beta power to the presence and nature of episodic bursts of beta activity. Here we test the hypothesis that beta bursts, though short in duration in more physiological state, may help explain spontaneous variations in motor behaviour of human adults at the single trial level. To this end we recorded local field potential activity from the subthalamic nucleus (STN) of Parkinsonian patients of both genders whose motor behaviour had been normalised as far as possible through treatment with the dopamine prodrug, levodopa. We found that beta bursts present in a time-limited window well before movement onset in the contralateral STN reduce the peak velocity of that movement and that this effect is further amplified by the amplitude of the burst. Additionally, prolonged reaction times are observed when bursts occur immediately after the GO cue. Together, these results suggest that the modulation of the timing and amplitude of beta bursts might serve to dynamically adapt motor performance. These results offer new insight in the pathology of Parkinson's disease, and suggest that beta bursts whose presence and nature are modulated by context may have a physiological role in modulating behaviour.

Keywords:

Beta oscillations; beta bursts; Parkinson's disease; motor performance; subthalamic nucleus; reaching movement.

Significant statement

Beta oscillations (~13-30Hz) have been increasingly interpreted as transient bursts rather than as rhythmically sustained oscillations (Feingold et al., 2015). Prolonged and increased probability of beta bursts in the subthalamic nucleus correlates with the severity of motor impairment in Parkinson's disease (Tinkhauser et al., 2017a,b). However it remains unclear whether beta bursts act to modify motor performance on a trial-by-trial basis under more physiological condition. Here, we found that according to the time window in which they fall, beta bursts reduced the velocity of the forthcoming movement or prolonged the reaction time. These results offer new insight in the pathology of Parkinson's disease and suggest that the modulation of beta bursts might serve to dynamically adapt motor performance.

Introduction

Neural oscillations in the beta frequency band (~13-30Hz) are a prominent feature in the cortico-basal ganglia motor network. During motor control, beta oscillations are systematically modulated showing a marked reduction of mean power prior to and during voluntary movement, followed by a rebound at the end of movement. This movement-related modulation of beta power has been observed in a multitude of motor tasks and in various cortical regions (Pfurtscheller & Lopes da Silva, 1999,; Tan et al., 2014a, 2016, Torrecillos et al., 2015, Fischer et al., 2016; see Kilavik et al., 2013 for a review), as well as in different structures of the basal ganglia (Cassidy et al., 2002; Kühn et al., 2004, Doyle et al., 2005, Tan et al., 2014b). Additionally, during tonic holding contractions cortical beta activity is coherent with the electromyogram of contralateral contracting muscles (Baker et al., 1997). Hence, beta oscillations in the cortico-basal ganglia motor circuit are now widely associated with motor control (Jenkinson & Brown, 2011, Singh et al., 2018).

More recently it has been realised that beta oscillations in this motor network emerge as brief transient events or bursts (Murthy and Fetz, 1992, 1996; Bartolo and Merchant, 2015; Feingold et al., 2015; Sherman et al., 2016; Tinkhauser et al., 2017a,b; Shin et al., 2017). Recordings in the subthalamic nucleus (STN) of untreated patients with Parkinson's disease (PD) at rest demonstrate that the mean duration of beta bursts is prolonged and that the probability of long beta bursts correlates with the severity of motor impairment (Tinkhauser et al., 2017b). This is likely to be related to the rise in burst amplitude, indicative of an increase in local neural synchronization, which negatively impacts upon the motor system when excessive (Brittain and Brown, 2014).

The change in beta power typically observed around movements has also been suggested to reflect changes in the probability of beta bursts rather than a smooth modulation of sustained beta activity (Feingold et al., 2015). Studies in non-human primates have confirmed that beta burst probability changes across trials with motor and cognitive processes (Feingold et al., 2015, Lundqvist et al., 2016). In patients with Parkinson's disease, the movement-related modulation in the beta band is reduced in the basal ganglia (Doyle et al, 2005) and the average beta desynchronization correlates with overall motor performance (Kühn et al, 2004). The reduced modulation in the beta power averaged over multiple trials may reflect impairment in the modulation of the timing of the beta bursts, suggesting that it is not only the duration of beta bursts but also their precise timing that can contribute to the motor impairment evident in Parkinson's disease. A recent study has demonstrated that the probability of cortical beta bursts before a stimulus can predict detection performance and attentional shifts in both animal and human data (Shin et al., 2017). However it is unknown how changes in the probability and timing of beta bursts around a go cue might affect motor performance.

Here, we test the hypothesis that the timing and amplitude of beta bursts in the basal ganglia modify motor behaviour by seeking predictive, within-subject correlations between beta bursts and motor performance in PD patients who have undergone surgery for deep brain stimulation and have been treated with the dopamine prodrug levodopa. These patients afford an opportunity to record local field potential (LFP) activity directly from the STN in the awake, behaving human. As patients were on medication, motor performance was optimised as far as possible and was tested in a visually cued joystick task, as measured by reaction time and movement velocity. We showed that the timing and the amplitude of beta bursts occurring in the contralateral STN before movement are associated with measurable changes in motor performance at the single trial level. According to the time window in which they fall, beta bursts can reduce the velocity of the forthcoming movement and/or slow down the reaction time.

Materials and methods

Subjects

Twelve patients (5 female) with Parkinson Disease gave their written informed consent to participate in the experiment, which was approved by the local ethics committees. Their mean age at the time of the recording was 63.8 years (range 56 to 70 years) with average disease duration of 10.8 years (range 4-17 years). All subjects were right handed by self-report and had normal or corrected-to-normal vision. Clinical severity was measured by using the Unified Parkinson's Disease Rating Scale and the mean score was 46.4 ± 4 in the OFF and 21.8 ± 2.7 in the ON medication state. Patients were implanted with deep-brain stimulation (DBS) electrodes (model 3389, Medtronic Neurological Division) in the left and right subthalamic nucleus (STN). The clinical details of the patients and of the surgical intervention are reported in Table 1.

Experimental Protocol

Subjects performed a visually cued joystick reaching task as described in Figure 1A. They were seated in front of a computer monitor and held a finger joystick with their right hand, which rested on a padded arm support. The position of the joystick was displayed on the computer monitor as a cursor in the form of a red circle with 6mm diameter. Subjects were instructed to make rapid out and back movements to move the cursor from the centre of the monitor to a target position. The target was a green circle (6mm diameter, 0.6 visual degrees) displayed on the screen. Each trial started with the red cursor in the centre of the monitor. Then a green target appeared at a position randomly selected from three positions equally spaced around an invisible arc with a radius of 7.5cm (6.1 visual degrees) and central angle of 90^{o}, which acted as the GO cue. The green target remained at its new position for 1 s before it disappeared. Subjects were instructed to respond as fast as possible after the GO cue by moving the cursor toward the green target in a ballistic and straight movement. To minimize any corrective movements, no visual feedback of the cursor position was provided during the movement. The position of the red cursor was presented at rest and disappeared after movement onset, once it had reached 5% of the maximal displacement. It reappeared once it had reached 90% of the maximal displacement to show the endpoint of the reaching movement. Thereafter the position of the red cursor did not respond to further corrective movements in that trial and returned to its central starting position when participants released the joystick. The cursor remained at the centre for 1.5-2s (uniformly distributed) before the next trial began, making the total inter-trial interval between 2.5 and 3sec. Note that in the present study the data from the three target positions were pooled and analysed together, as a visual inspection of the hand paths and velocity profiles revealed no systematic difference between the three directions. After familiarization with the apparatus, each subject performed

50 trials that corresponded to the baseline session of a longer experiment (not described here).

Data recording

Recordings were made when the patients were on their usual dopaminergic medication, between 3 and 6 days postoperatively, while electrode leads were still externalized and before implantation of the pulse generator. STN local field potentials (LFPs) were recorded from the four different contacts of each implanted electrodes (right and left STN) using a 32-channel TMSi-Porti amplifier and its respective software (TMS International, Netherlands). The ground electrode was placed on the left forearm. LFP signals were amplified, low-pass filtered at 550 Hz, sampled at 2048Hz and common average referenced. The behavioural task was presented using open-source software (PsychoPy version 1.74). To synchronise the behavioural measurements and the LFP recordings, a trigger signal was generated using PsychoPy software and converted to an analogue signal through a digital-to-analog converter (U3; LabJack). This trigger signal changed from 0 to 3V at the start of each trial and was simultaneously recorded with the monopolar LFPs using the same amplifier (TMSi). The displacement of the joystick in x and y axes and the timing of the target jump were also recorded through the TMSi-Porti amplifier and sampled at 2048 Hz.

Behavioural analysis

Behavioural data were analysed off-line using custom-written MATLAB scripts (version R2015b; MathWorks). The position of the cursor was differentiated to calculate velocity, which was subsequently filtered through a Gaussian kernel with a window duration of 10 ms. As illustrated in Figure 1B, the joystick velocity profiles were characterized by two distinct

peaks corresponding to the reaching movement (center-out) followed by the joystick release (center-in), respectively. To assess the motor performances of each subject we focused our analysis on two main behavioural parameters; the reaction time and the velocity peak of the outgoing movement. First, we defined the movement onset of each single movement as the time when the joystick velocity crossed the threshold of three times the standard deviation of the signal (and its noise) at rest, and sustained this speed for at least 100ms. The reaction time was then computed as the delay between the GO cue and the movement onset (RT, see inset of Fig 1B). Second, the amplitude of the velocity peak of the out reaching movement was defined for each trial (VelPA, see inset of Fig 1B). For both the coefficients of variation were computed for each subject by dividing the standard deviation by the mean and multiplying by 100.

Due to the high kinematic variability between and within subjects (see for instance Fig 1B and 1D), the velocity profiles of all individual trials were visually inspected to manually correct movement onset and peak velocity when necessary. For further analyses, trials with extra-long reaction time (more than mean 2.5 SD) were discarded. Similarly, trials with abnormal hand path trajectories or in which the hand was not maintained stable enough during the inter-trial interval were visually identified and excluded.

STN-LFP pre-processing

All LFP data pre-processing were performed offline using the free and open-source Fieldtrip toolbox (Oostenveld et al. 2011). Before any analysis, LFP recordings were down sampled to 1000 Hz and bandpass filtered between 1 and 100 Hz. Continuous time series were segmented into 4 seconds epochs, from -1.5s until 2.5s after the GO cue or the movement onset. Note that continuous time series were also processed as described below to determine

the mean characteristics of bursts (duration and amplitude, see Results). Individual trials were visually inspected, and those with channels containing artefacts were excluded. LFP signals were then converted to bipolar montages between adjacent contact pairs resulting in three bipolar montages per STN to limit the effects of volume conduction from distant sources (Marmor et al., 2017). After behavioural and electrophysiological artefact removal, analyses were based on averages of 42.4 ±1.5 trials by subject, resulting in a total number of 506 included trials.

LFP analysis: Frequency–time decomposition, channels and beta peak selection

Single-trial LFP signals were transformed in the time-frequency domain by convolution with complex Morlet wavelets characterized by the ratio $f0/\sigma f = 7$, with f0 ranging from 1 to 45Hz by steps of 0.25Hz. Event-related changes in power were calculated by normalizing for each frequency band the value of each time point against the mean power calculated across all trials. For each subject, the normalized power was separately averaged over all trials for each of the three bipolar contacts for each STN. The bipolar contact with the largest movement-related power change in the whole beta band (13–30 Hz), i.e., the largest difference between the trough of the event-related desynchronization (ERD) during movement and the peak post-movement synchronization (ERS) in the beta band, was then selected for further analysis. This was motivated by evidence linking maximal beta band activity to the dorsal (motor) region of the STN (Chen et al., 2006; Zaidel et al., 2010; Horn et al., 2017) and maximal beta band movement-reactivity to the site that offers the most effective deep brain stimulation (Ince et al., 2010; Zaidel et al., 2010; Tinkhauser et al., 2018), this site corresponding also to the one with the maximal beta band movement-reactivity (Devos et al., 2006).

For each chosen bipolar contact pair the beta frequency peaks were individually selected. To this end, the movement-related beta power modulation was computed across all trials for each beta frequency (from 13 to 30Hz in 1Hz steps). The frequency with the largest difference between ERD and ERS was then selected. Time-frequency maps and normalized beta power time-courses were also visually inspected to confirm the contact and frequency peak selection. Across all subjects, this selection process results in a mean frequency of 19.6Hz ±1.3Hz for the left STN and 18.7Hz ±1.1Hz for the right STN.

LFP analysis: bursts detection

To explore the trial-by-trial relationship between beta oscillations and motor performance we used the concept of beta bursts (Tinkhauser et al, 2017a, b). Beta bursts were detected according to the following procedure. First, beta power time courses were computed for each single trial by averaging over a 6Hz-wide frequency band centred on the contact's beta peak frequency (see above, Fig. 2B). A threshold was set at the 75th percentile of the mean beta power calculated for each subject and STN over the individualised beta frequency band across the whole session. Note that in contrast to Tinkhauser et al. (2017 a, b), the thresholds were defined based on data including cued movements. All time points surpassing the threshold were labelled as "potential bursts" and only those lasting more than 2 oscillatory cycles were definitively defined as "beta bursts" (Fig. 2C). Thus, the minimal beta burst duration depended on the individual frequency band and was different for each subject. Across subjects, the minimum burst duration was on average 111ms ±7ms for both STN (ranging from 73ms to 163ms). The probability of bursts was computed as the number of burst trials divided by the total number of trials for each subject. The impact of the burst detection threshold was also tested by using eight different thresholds ranging from 50% to

220 85% in steps of 5% (Fig. 3B or C). Note that the threshold couldn't be increased further as
221 too few trials with bursts were detected with a 90% threshold.

222

223 *LFP analysis: extraction of bursts features*

224 To determine the influence of STN bursting activity on motor performances we first
225 considered a window from -600ms to the GO cue (Fig 1A). Based on the beta power profiles
226 and the mean inter-trial interval, the duration of the window was set to 600ms to avoid any
227 overlap with the end of the previous trial and ensure that beta rebound of that previous
228 movement was excluded. On average, across subject, the delay between the end of the last
229 movement and the GO cue was 1.88 ± 0.07 sec. For each subject and STN the number of
230 bursts in the window was calculated by keeping only bursts with more than half of their
231 duration in the window. This meant that some bursts could overlap with the presentation of
232 the GO cue. Each trial with at least one burst in the window was labelled as "burst trial". All
233 other trials were labelled as "no-burst trials".

234 To characterize the impact of bursts on the next movement we then extracted their main
235 features: amplitude, duration and timing. For trials with more than one burst before and/or
236 overlapping with the GO cue only the last burst was considered. The burst amplitude was
237 calculated by averaging the power value of each time point exceeding the burst detection
238 threshold of 75^{th} percentile. The burst timing corresponded to the time between the
239 termination point of the beta burst and the GO cue. Importantly, the timing could be negative
240 if the termination point occurred before the GO cue, or positive if it occurred after the GO
241 cue.

The effect of the timing of bursts was further explored by testing the impact of the presence of bursts in short time windows of 50ms (bins). Based on our results, bins were defined relative to the GO cue from -400ms to +200ms. The bin [+200ms:+250ms] was not included due to the small number of bursts observed for some subjects (less than 3 bursts for 3 subjects) due to the typical pre-movement beta desynchronization (Fig. 2). For each bin, each single trial was labelled with a "1" if at least one time point of the bin exceeded the burst detection criteria.

Bursts in lower and higher frequency bands

To confirm the specificity of effects to the beta band, similar analyses were performed in two other frequency ranges: the theta/alpha range and the low gamma range. For both, bursts were defined in a 6Hz band derived by shifting the individually defined beta peak frequency up or down. The low gamma range was derived in each subject by adding 20Hz to the frequency of their beta peak. This avoided any overlap with the high beta band (lower limit of the low gamma range >30Hz in all subjects). Across subjects the selected mean low gamma frequency band was centred on 39.6 ±1.3Hz. For the theta/alpha range we could not systematically subtract the same number from each individual's beta peak frequency as this resulted in low frequency peaks ranging from the delta to the low beta range. Thus, to avoid this heterogeneity and constrain all the frequency peaks in the alpha range, the same frequency band was considered for each subject (8-12 Hz). Then all bursts analyses were performed as previously described for the beta band.

Statistical analysis

Statistical analyses were performed using the free software R (v3.3.1). We used the *nlme* package (Pinheiro et al., 2018) to perform linear mixed effects models of the single-trial relationship between beta oscillations and behavioural performances. To correct the non-normality of the dependent variables, the reactions times were log-transformed and the peak velocities were raised by the lambda exponents identified by a box-cox procedure (power transformation). The normal distribution of each variable was then visually inspected with quantile-quantile plots and histograms of distribution. All models were estimated by the method of maximum likelihood and included random intercept for subjects, to allow different intercepts for each subject capturing individual differences.

To explore the effect of bursts that had more than half of their duration in the 600ms time window before the GO cue we first defined the presence of a burst (trials labelled with 1 or 0) as fixed effect and tested its impact on each behavioural parameter separately (RT and VelPA). Second, if the presence of a burst had a significant impact on a motor parameter, we performed a new linear mixed effect analysis to evaluate the influence of the burst features. To this end we entered each burst feature separately (burst amplitude, duration and timing) as individual factors. When multiple features significantly contributed to the prediction, but were correlated to each other, the different models were compared based on the Akaike's Information Criterion (AIC) and the correlation between the predicted and actual measured values (r^2). If the predictors were not correlated, a model including all significant factors was compared to the model that included only one factor to assess whether the model's improved fit to the data merited the added complexity associated with the inclusion of that component (likelihood ratio test).

For the binning procedure, linear mixed-effect models were estimated with the presence of a burst in each bin as fixed factor and the velocity peak or the reaction time as dependant variables. For all models the residuals plots were visually inspected to control for any obvious deviation from homoscedasticity or normality. Multiple comparisons were corrected for using the false discovery rate procedure (Benjamini & Hochberg, 1995).

Results

In the present study our principal goal was to explore the within-subject relationship between transient beta oscillations and motor performance in treated PD patients. To do so we performed single-trial analysis by focussing on the effects of pre-movement beta bursts on two motor parameters: the reaction time and the peak velocity.

Behavioural results

Subjects performed 50 reaching movements by controlling a joystick with their right hand to move a red cursor from a starting position in the centre of the monitor to one of three green targets displayed on the screen (see Figure 1A). They were instructed to respond as fast as possible after the GO cue (target appearance) and to perform ballistic movements. The velocity profiles were two-peaked with the first peak corresponding to the outgoing movement and the second one to the joystick release, which resulted in the cursor returning to the centre (Fig. 1B). For each single trial, the reaction time and the peak velocity of the outgoing movement were extracted (see insert of Fig. 1B). These were averaged across trials for each subject and then averaged across subjects. Mean reaction time and peak velocity were 413 ± 21ms (314 – 533ms, Fig. 1E) and 0.27 ± 0.02 m/s (0.14 - 0.4 m/s, Fig. 1C), respectively. These behavioural results based on subject averaged data reflect the inter-

312 subject variability but ignore the trial-by-trial variability in behaviour that may or may not be
313 linked to the dynamics of beta oscillations in the STN. The within-subject variability is
314 illustrated in Figure 1D and can be quantified by the coefficient of variation, computed for
315 each subject across trials. Across subjects, the coefficient of variation for the reaction time
316 was 20.7 ± 1% (14-28%, Fig. 1E), and 22.4 ± 1.9% for the peak velocity (14-40%, Fig. 1C).
317

318 **Beta burst characteristics**

319 As illustrated in Figure 2A, beta bursts were defined as beta amplitude exceeding the 75th
320 percentile threshold of beta power in a 6Hz frequency band centred on the individual beta
321 frequency peak (see Methods). Across all subjects, the mean burst frequency was centred on
322 19.6±1.3Hz for the left STN and 18.7 ±1.1Hz for the right STN. The mean duration of beta
323 bursts across subjects was 207.6 ± 16.2ms and their mean amplitude was 1.45 ± 0.04 au (see
324 Fig. 2C). The mean burst duration is similar to the burst duration previously reported in PD
325 patients ON medication, in contrast to the longer bursts observed OFF medication (274ms
326 and 406ms respectively in Tinkhauser et al., 2017b). Note that the slight difference between
327 our results and this previous report might be due to the smoothing of the LFP signals applied
328 in the latter (0.2sec in Tinkhauser et al., 2017b). On average, bursts longer than 600ms, which
329 have been previously correlated with clinical impairment in PD patients (Tinkhauser et al.,
330 2017a, b), comprised 6.1±3.2 % of the total burst time and 2.2±1 % of total number of beta
331 bursts. The amplitude of beta bursts increased with burst duration, with a significant positive
332 correlation observed for all the subjects ($p<0.05$, $r = 0.42 \pm 0.04$ across subject, see Fig2.C
333 and Fig. 2B for one example subject)
334

Presence of beta bursts before and overlapping the GO cue reduces the peak velocity of the following movement

The first question we asked was whether the presence of beta bursts before the GO cue affects the following movement. To this end, bursts were considered in a temporal window beginning 600ms before the GO cue to avoid inclusion of the beta rebound typically observed at the end of the last movement. Across subjects the mean delay between the end of the last movement and the GO cue was 1.88 ± 0.07 sec. We included bursts with more than half of their duration in the 600ms time window, which meant that some bursts could overlap the presentation of the GO cue. Across all subjects, at least one burst was observed in the window for 60 ± 4% of all trials. Trials with a burst were labelled with a '1' (300 burst trials across all subjects) and trials without any burst with a '0' (206 no burst trials). To explore the impact of bursts on motor performance within each subject, we performed linear mixed-effects analyses with fixed effects describing the relationship between the presence of a burst and each of the two movement parameters separately (reaction time and peak velocity).

The presence of a burst in the 600ms window before the GO cue resulted in a significant difference in the peak velocity of the next movement (b = -0.0135, $t_{(493)}$ = -2.4, p=0.016, Table 2). The direction of the relationship (b<0) indicated that trials with bursts in this window were associated with lower velocities. To corroborate and visualise this effect, average peak velocities of trials in which bursts occurred (normalized to all trials) were plotted for each subject (Figure 3A). The effect with velocity was selective so the presence of a burst in this time window did not affect reaction time (p=0.31). Moreover, the relationship between peak velocity and burst occurrence was confined to the STN contralateral to the active limb, since the model with ipsilateral beta bursts was not significant (p=0.75). The relationship with velocity was maintained irrespective of whether bursts in the contralateral

360 STN were defined with a 75th or 80th percentile threshold (80th; b = -0.014 $t_{(493)}$ = -2.4,
361 p=0.02, Fig. 3C). Hereafter, we limit further analysis to bursts determined using our default
362 75th percentile threshold.
363

364 **Amplitude of the burst before or overlapping the GO cue also reduces the velocity of the**
365 **following movement**

366 The fact that the peak velocity was slower when preceded by bursts, defined as beta power
367 exceeding a high threshold, raises the possibility that the amplitude of episodes of beta
368 activity matters. This hypothesis was further supported by the greater peak velocity reduction
369 when higher thresholds were used to define bursts (Figure 3B). Accordingly we specifically
370 tested if, when a burst occurs, its amplitude further influences velocity in the following
371 movement. To deal with trials for which more than one burst was found in the pre-GO time
372 window, we only considered the last beta burst in the window (the burst closest to the GO
373 cue). Note that where more than one burst occurred within the window of interest (29% of
374 trials) the last bursts were no different in amplitude to earlier bursts ($t_{(10)}$=0.09, p=0.9). Our
375 model confirmed that higher amplitude beta bursts before or overlapping the GO cue were
376 associated with a lower peak velocity in the following movement (b = -0.01, $t_{(493)}$ = -3.2,
377 p=0.0015). The effect was again specific for the contralateral STN (ipsilateral STN, p=0.78)
378 and for the velocity peak (reaction time, p=0.11). To illustrate the relationship between burst
379 amplitude and peak velocity, Figure 4 shows scatterplots from each subject.
380

381 Critically, we also confirmed that the effect was specific to burst amplitude, and not
382 secondary to the mean beta power over the same 600ms window in each trial. Whereas a
383 similar relationship between mean power and velocity could be observed when all trials were

included in the model (506 trials, b = -0.013, $t_{(493)}$ = -2.2, p=0.03), the model was no longer significant after FDR correction (p corrected =0.06, Table 2). In addition, a model that only considered beta power in no-burst trials was not significant (206 trials, 17±1.7 trials per subject; $t_{(193)}$ = 0.13, p=0.9). This result suggested that sub-threshold beta power (< 75th percentile amplitude) does not contribute to the behavioural outcome. In contrast, the last burst amplitude still predicted the velocity when only burst trials were entered in the model (300 trials; 25±1.8 trials per subject; b = -0.013, $t_{(287)}$ = -2.5, p=0.014, Table 2).

In addition to the burst amplitude we also extracted the duration of the last burst before the GO cue, which was highly correlated with the burst amplitude (r=0.77, p<0.001 across all trials). As an individual factor, the burst duration revealed a weak relationship with the peak velocity (b = -0.005, $t_{(493)}$ = -2.1, p=0.04), which, however, did not survive multiple comparisons corrections (corrected p = 0.07). This weaker relationship might be explained by the smaller range of burst duration as compared to the range of burst amplitude (Fig. 2C).

When is motor performance most vulnerable to beta bursts?

To explore when precisely velocity was most affected by the occurrence of a beta burst, we next considered their timing. To this end, we defined the timing of the last burst beginning before the GO cue as the delay between its termination point and the GO cue. Importantly, this termination point could occur before (negative delay) or after the GO cue (positive delay). There was a clear relationship between the termination of the last burst before the GO cue and the reduction of velocity peak (b = -0.031, $t_{(493)}$ = -2.8, p=0.006, Table 2) whereby bursts ending close to or shortly after the GO cue were more likely to slow down movement velocity.

These results suggest a limited window in which bursts affect movement velocity. To test this hypothesis further we considered the effect of bursts in bins of 50ms duration around the GO cue. As can be seen in Figure 2, the post-GO cue window corresponds to the time period in which the pre-movement beta desynchronization is typically observed. Hence, the probability of a burst drops rapidly to reach its minimum around the movement onset. We therefore considered twelve bins from -400ms to +200ms around the GO cue and stopped at +200ms as this was the end of the last bin [+150ms:+200ms] where bursts were present in at least 3 trials for each subject. The number of burst trials per bin comprised between 83 ([+150:+200ms]; 7±0.8 per subject) and 135 trials ([-400:-350ms], 11.3 ± 1 per subject). The results confirmed the timing effect and revealed three significant bins around the GO cue (b = -0.014 $t_{(493)}$ = -2.2, p=0.032; b = -0.015, $t_{(493)}$ = -2.1, p=0.035; b = -0.016, $t_{(493)}$ = -2.4, p=0.018, for the three bins, respectively) which, however, did not survive multiple comparisons corrections (Fig. 5A). Yet, these results suggest that bursts had to terminate just before or after the GO cue to have an effect on the peak velocity of the following movement. They also had to occur in the contralateral STN, as the same binning procedure revealed that bursts in the ipsilateral STN failed to correlate with velocity (p>0.05 for all bins).

Based on these results, however, the lack of effect previously observed for the subthreshold mean beta power over the 600ms pre-GO window could in fact be due to the size of the time window that excluded power at and just after the GO cue, and did not allow for a differential effect closer to the GO cue. Therefore to confirm the selective effect of bursting we also tested the relationship between velocity peak and mean beta power in each of the 12 time bins around the GO cue. When keeping all trials, four significant bins were observed from -200ms to the GO cue (b = -0.005, $t_{(493)}$ =-2.1, p=0.037; b = -0.007, $t_{(493)}$ = -2.6, p=0.009; b = -0.008, $t_{(493)}$ = -2.5, p=0.014; b = -0.007, $t_{(493)}$ = -2.2, p=0.032 for the four bins, respectively), but as

433 for the presence of a burst, none were still significant after FDR correction. Moreover, when
434 removing the trials with bursts the subthreshold mean power failed to predict the velocity
435 peak ($p>0.05$ for all bins). It was unlikely that this absence of relationship with beta power
436 was related to small sample size as the number of no burst trials by subject was on average
437 between 32±2 and 35.5±1.8 for each bin (i.e. ≥3 times the number of burst trials).
438
439 The same binning procedure was then applied with bins defined relative to the Movement
440 Onset, and the results revealed a larger critical window with three significant bins after
441 multiple comparisons corrections (Fig 5B, b = -0.019, $t_{(493)}$ =-3, p=0.003; b = -0.024, $t_{(493)}$ =
442 -3.7, $p<0.001$; b = -0.02, $t_{(493)}$ = -3.2, p=0.001; for the three bins, respectively). The bin [-
443 500:-450ms] was significant when considered in isolation (b = -0.015, $t_{(493)}$ = -2.2, p=0.03)
444 but not after multiple comparisons corrections. This result and the bigger estimated effects
445 observed for the Movement Onset alignment compared to GO cue alignment (see Fig.5A and
446 B) suggest that bursts had to fall around 650 to 500ms before the movement to impact
447 velocity. Considering the reaction times (Fig.1E) these same bursts might therefore overlap
448 with the GO cue when trials were aligned to the latter, although here the relationship was
449 weaker (Fig 5A). To clarify this we determined the end points of the beta bursts occurring in
450 the whole significant window aligned to the movement onset (blue shading in Fig. 5B). The
451 results revealed that most of them occurred before the GO (end point before the GO or
452 shortly after, sign-rank test, Z=78, $p<0.001$, Fig 5.C).
453
454 In summary, beta bursts present in the contralateral STN just before or around the time of the
455 GO cue reduced the peak velocity of the subsequent movement. This effect was likely
456 secondary to the timing of these bursts with respect to the movement itself. The biggest effect
457 of beta bursts on velocity was observed when these were aligned to movement onset and not

458 GO cue presentation. Of note, this effect of beta bursts falling around 650 to 500ms before
459 movement onset was time-limited, and bursts occurring after this, but still before movement
460 onset, had no significant effect on velocity (Fig 5B).
461

462 **Bursting after the GO cue affects reaction time**

463 The binning procedure reported above was repeated for reaction time and revealed significant
464 effects of the presence of beta bursts upon reaction times in all four bins after the GO cue
465 (Fig. 6A, b = 0.06, $t_{(493)}$ = 2.5, p=0.01; b = 0.09, $t_{(493)}$ = 3.4, p<0.001; b = 0.08, $t_{(493)}$ = 3.3,
466 p=0.001; b = 0.07, $t_{(493)}$ = 2.8, p=0.005 for the four bins respectively). Reaction times were
467 longer in trials in which beta bursts were present in the 200ms after the GO signal (Fig 6B).
468 These results are in line with the significant relationship observed between the timing of
469 bursts in the pre-GO window and the reaction time (b = 9.80E-05, $t_{(493)}$ = 2.4, p=0.02; Table
470 2), which suggested that bursts had to end after the GO cue to affect the reaction time. This
471 effect was again confined to the contralateral STN (ipsilateral STN p>0.05 for all bins). To
472 confirm the selective effect of bursting we also tested the relationship between reaction time
473 and mean beta power in each bin. When all trials were included, the three bins from 50ms to
474 200ms showed a significant effect (b = 0.03, $t_{(493)}$ = 2.5, p=0.012; b = 0.03, $t_{(493)}$ = 2.9,
475 p=0.004; b = 0.02, $t_{(493)}$ = 2.03, p=0.04, for the 3 bins respectively), which disappeared after
476 multiple comparison corrections and when only trials without bursts were considered.
477

478 We also tested the effect of bursts when the bins were aligned to the Movement Onset. In
479 contrast to the bursting effect on velocity, the effect on reaction time was then no longer
480 observed (Fig. 6C, p>0.05 for all bins). Thus, the effect of bursts on reaction time was
481 determined by their precise timing with respect to the GO cue, and not, unlike the effect on

velocity, on the timing with respect to movement onset. Still, the presence of bursts several 100ms before movement onset already reflected differences in reaction time. This effect was also time-limited, as the probability of bursts dramatically reduced soon after the GO cue (Fig. 2A).

Effects of bursts on motor performances are confined to the beta band

To test the specificity of the described effects to the beta band we tested the impact of bursting activity on motor performance in two other frequency bands. The first was the alpha frequency range with a similar 8-12Hz frequency band considered for each subject, and therefore sparing the lower beta band. Activity in the alpha band was again thresholded at the 75^{th} percentile. The mean duration of bursts in this band was 342.3 ± 4.8ms, and as for beta bursts, the amplitude of the alpha bursts increased with the burst duration ($p<0.05$ for all subjects, across subject $r=0.37$). However, the presence of an alpha burst in the contralateral STN before or overlapping with the GO cue was not significantly related to the motor performance (155 bursts trials, $p>0.05$ for both velocity and reaction time).

The second frequency band was in the low gamma range and was derived by adding 20 Hz to the frequency of the beta peak in each subject. The 6Hz band was centred on 39.6 ± 1.3Hz, and again did not overlap with the beta band (>30Hz for all subjects). The mean duration of low gamma bursts was 86.2 ± 2.4ms and, as for the alpha and beta bursts, significantly increased with the burst amplitude ($p<0.05$ for all subjects, across subject $r=0.3$). The linear mixed effect analysis revealed no significant relationship between the low gamma bursts in the contralateral STN before and overlapping the GO cue and the motor performance (415 bursts trials, $p>0.05$ for both the velocity and the reaction time). Together, these results

indicate that the effects of bursts on both the velocity and the reaction time were specific to the beta frequency band.

Discussion

Our results showed that, in treated PD patients, STN beta bursts occurring before movement are associated with measurable changes in motor performance within subjects. First, beta bursts present in a time-limited window around the GO cue reduce the peak velocity of the subsequent movement and this effect is further amplified by the amplitude of the burst. Second, beta bursts present immediately after the GO cue increase the reaction time. Importantly, we confirmed that the variations in motor performance were better explained by the beta bursts than averaged beta power and that effect of bursts, were limited to the STN contralateral to the active limb and confined to the beta frequency band.

Beta bursts ON medication are briefer than OFF medication

The transient nature of beta oscillations is now well established and observed at both the cortical (Feingold et al., 2015; Lundqvist et al., 2016; Sherman et al., 2016; Shin et al., 2017) and subcortical level (Bartolo and Merchant, 2015; Feingold et al., 2015). The duration of beta bursts may serve to distinguish pathological from physiological beta activity in patients with PD (Tinkhauser et al., 2017a, b). Beta bursts are more often longer in untreated patients compared to ON medication, and the increased probability of bursts longer than 600ms positively correlates with clinical impairment. For instance, OFF medication, 40% of the total burst duration and 20% of the total number of defined bursts were longer than 600ms (Tinkhauser et al., 2017a). This compares with 6% of the total burst duration and 2% of the

529 total number of bursts in the present study where patients were ON medication. Our results
530 show that beta bursts, even when of short duration, can also affect motor performance when
531 they happen in a specific time window relative to the movement. These findings lead us to
532 posit that the predominant brevity of beta bursts could be important in normal beta-band
533 function (Feingold et al., 2015; Lundqvist et al., 2016; Shin et al., 2017).
534

535 **Beta bursts and their timing predict behavioural dynamics**

536 According to the time window in which they fall, beta bursts in the contralateral STN were
537 associated with reduction of movement velocity or prolongation of reaction times. These
538 results add to the growing evidence that elevated beta oscillations are linked to slowing of
539 movement.

540 Clinical observations have related gross movement slowing, termed bradykinesia, to
541 exaggerated oscillatory beta band synchronization (Kühn et al., 2006; Ray et al., 2008) and to
542 longer and higher amplitude beta bursts (Tinkhauser et al., 2017a,b). In PD patients, STN
543 stimulation at 20Hz reduced movement velocity in a tapping task (Chen et al., 2007) and
544 contraction velocity in a gripping task (Chen et al., 2011). Similarly, transcranial alternating
545 current stimulation at 20Hz applied over the motor cortex of healthy participants slowed
546 down the initial and peak velocity of voluntary movements (Pogosyan et al., 2009).

547 The prolongation of reaction time associated with beta bursts present just after the GO cue is
548 consistent with previous results showing that short latencies of the pre-movement
549 desynchronization in STN beta power are associated with short reaction times across PD
550 patients (Kühn et al., 2004) and even across single trials within individual subjects,
551 independent of the medication state (Williams et al., 2005). This is in line with the
552 observation that high-amplitude beta activities in motor cortical regions during critical

553 preparatory periods delay movement onset in non-human primates performing a
554 neurofeedback reaching task (Khanna and Carmena, 2017) or in healthy participants
555 performing joystick tasks (Boulay et al., 2011, McFarland et al., 2015).

556

557 **Time-dependant effects of beta bursts**

558 Consistent with previous findings, our results demonstrate that beta bursts relate to
559 differences in motor performance way beyond their termination (Gilbertson et al., 2005,
560 Androulidakis et al., 2007, Herz et al., 2018). For example, Shin et al 2017 found that beta
561 bursts have an effect on detection/attentional performances that outlasted their duration by
562 ~200ms. Our results suggest that the impact of bursts upon function strongly depends on the
563 time window in which they fall relative to the movements, presumably because processing
564 related to different functions dominates in different time windows throughout a task. The
565 effect of beta bursts on reaction time was observed immediately following the GO cue, which
566 informs the subjects about the direction of the reach. This information may be contrasted with
567 evidence drawn from earlier trials about the probabilities of targets, given only three options
568 were available. Where expectations and instructions do not coincide it may be advantageous
569 to delay responses to avoid wrong prepotent responses. A time-limited delaying effect of beta
570 bursts has also been reported in the STN of untreated PD patients in a brief post-GO cue time
571 window (~100ms) in the setting of more explicitly conflicting information (Herz et al., 2018).
572 The latter, together with the trial-by-trial relationship between cortical beta bursts and
573 detection performance reported by Shin et al., (2017), also suggests that beta synchrony is not
574 exclusively motoric in its consequences (Engel and Fries, 2010).

575

In contrast to the effect on reaction time, beta bursts affecting movement velocity were better aligned to movement onset than to the GO cue. Surprisingly, most of these bursts already terminated before the target was specified (GO-cue). As response vigour is not necessarily dependent on the response direction, it could be determined prior to the GO cue, particularly when the little variation in the timing of trials allows temporal expectancy, as in our paradigm. Accordingly, beta bursts before the GO cue may impact the specification of the movement vigour, previously associated with the STN (Turner and Desmurget, 2010). Thus movement triggered during periods of elevated beta synchrony (i.e with bursts estimated by finger microtremor) are slowed compared to movements that are randomly triggered, and a negative correlation between bursts of cortical synchrony and response acceleration may similarly occur around or before the cue (Gilbertson et al., 2005).

Here we showed that brief episodes of over synchronisation, as quantified by beta bursts, explained variations in behaviour better than averaged beta power before movements. By identifying the precise time window relative to movements in which the presence of beta burst can have a modulatory effect on the motor performance, our results offer new insights on the pathology of Parkinson's disease. The lack of modulation in the timing of beta bursts relative to movement may contribute to reduced movement-related desynchronization previously observed in averaged data (Doyle et al, 2005).

Beta bursts may have functional significance through excessive synchronisation

In the above discussion we have assumed that bursts can be considered discrete events whose impact on motor performance increases with amplitude above a threshold value. The alternative is that instantaneous beta amplitude impacts on motor performance as a

600 continuous, linear variable, with threshold crossings merely representing stochastic
601 deviations in a random signal. The present study alone cannot categorically distinguish
602 between these two possibilities, although the lack of an effect of instantaneous beta amplitude
603 in trials without suprathreshold activity (i.e bursts) in the critical time-windows would be
604 more in favour of the former interpretation. Additionally, the previously reported frequency-
605 selective temporal overlapping of beta bursts and phase synchronisation between sites that
606 respectively exceed that expected by chance and that present in non-burst periods also serves
607 to suggest that beta bursts may have a special significance (Tinkhauser et al., 2017a,b;
608 2018b).
609 How might a non-linearity arise to underpin the behavioural associations confined to high
610 amplitude bursts? Here it should be noted that the amplitude of LFP activity in the beta band
611 is a proxy for the degree of local synchronisation of neural elements in this frequency band.
612 Synchronisation is often viewed as advantageous as it increases the signal-to-noise ratio of
613 neural communication (Hanslmayr et al., 2012; Brittain and Brown, 2014). However, as
614 synchronisation increases, this effect will eventually be offset by the inherent restriction in
615 information coding capacity of the circuit entailed by synchronisation across its elements
616 (Mallet et al., 2008; Brittain and Brown, 2014). At that point, ever increasing synchronisation
617 may have an increasingly negative effect on the performance of the circuit. We speculate that
618 it is the crossing of this point that leads to the behavioural associations of bursts demonstrated
619 here. This however, does not necessarily mean that such behavioural effects are uniformly
620 deleterious. Brief increases in beta activity in the STN have been linked to the beneficial
621 delaying of responses in the presence of conflicting information (Herz et al, 2018). Thus there
622 may be contexts in which the dynamic control of network performance by varying beta
623 synchrony might represent a means of adjusting behaviour according to context on a trial-by-
624 trial basis (Feingold et al, 2015). Intriguingly, the impaired event-related desynchronization

reported in PD patients OFF medication implies that the occurrence of beta bursts may be less modulated by movements when dopaminergic activity is diminished (Doyle et al, 2005). Taking these observations together, we posit that beta bursts whose presence, size and duration are modulated by context may have a physiological role, but that this modulation may fail in untreated Parkinson's disease. Further studies are warranted to test and explore this framework.

Limitations

The present study was performed in patients with Parkinson's disease therefore it remains uncertain whether our findings apply to healthy participants in whom such intracranial LFPs cannot be recorded. The patients we studies were ON medication and were able to perform the task without any observable impairment. Analysis of group data confirmed that they have similar reaction times to healthy volunteers performing the exact same task (sign-rank test, $p=0.38$), but did indicate that patients' movements were significantly slower (sign-rank test, $p<0.001$). Overall, a key unanswered question remains whether the correlations observed here between STN beta bursts and motor performance reflect a physiological neural correlate of reaching behaviour or are linked to the underlying pathology.

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Figure Legends

Figure 1: Task and behavioural results. A. Visual stimuli in the joystick task and timeline of each trial. Single trial beta oscillations were analysed in the pre-movement period, from -600ms before the GO cue to -200ms before Movement Onset (yellow shading). The dashed circle outlines were not visible to the subject. During movement, only the endpoint feedback of the red cursor position was shown. **B.** Velocity profiles averaged across all trials for each subject (grey) and the grand average computed across all subjects (black). The time is normalized between two consecutive GO cues (100%) to average trials of different duration. The inset illustrates how the reaction time (RT) and the amplitude of the velocity peak (VelPA) were defined for each trial. **C.** Mean peak velocity of each subject and their coefficient of variation (CV) **D.** Velocity profiles of all individual trials and all subjects (n=506 trials, 12 subjects) relative to the GO cue. **E.** Mean reaction times of each subject and their coefficient of variation (CV)

Figure 2: Definition of beta bursts. A. Single trial data for one subject sorted by reaction times. The beta power time courses were computed by averaging over a 6Hz frequency band centred on the individual beta frequency peak. Then bursts were defined as beta amplitude exceeding the 75th percentile threshold with a minimum duration of 2 cycles. The black and red dots indicate the GO cue and the Movement onset respectively. **B.** Positive correlation between the burst duration and amplitude in one example subject (same as for A.; $r=0.56$ $p<0.001$). **C.** Mean burst duration and amplitude and positive correlations between the two for the twelve subjects. For all plots only the contralateral STN was considered.

Figure 3: Effect of bursts before and overlapping with the GO cue on the amplitude of the peak velocity and impact of burst detection threshold. A. Mean peak velocity in burst trials normalized (z-score) to the mean velocity of all trials for all subjects. A negative value indicates a reduction of peak velocity in burst trials. Trials are divided according to the presence of a burst in a 600ms window before the GO cue where bursts are only included if more than half of their duration falls in the time window. Bursts were defined with the default threshold of 75th percentile. **B.** Impact of burst detection threshold on the peak velocity reduction. For each subject the velocity peak of each trial is normalized (z-scores) as

described for A. **C.** Estimated effects and 95% confidence intervals derived from the linear mixed-effects models testing the impact of bursts occurring before or overlapping with the GO cue on peak velocity. Burst detection thresholds stop at 85th as too few trials with bursts were identified for the next 90th threshold. Note that for the modelling the peak velocities were power transformed (see Methods). * = significant model, $p<0.05$.

Figure 4: Single trial data in individual subjects illustrating the relationship between last burst amplitude and peak velocity. The linear mixed-effects model showed a negative relationship between the amplitude of the last burst before or overlapping the GO cue, and the peak velocity (25±1.8 burst trials per subject; b = -0.013, $t_{(287)}$ = -2.5, p=0.014). Note that only the burst trials of the contralateral STN are considered.

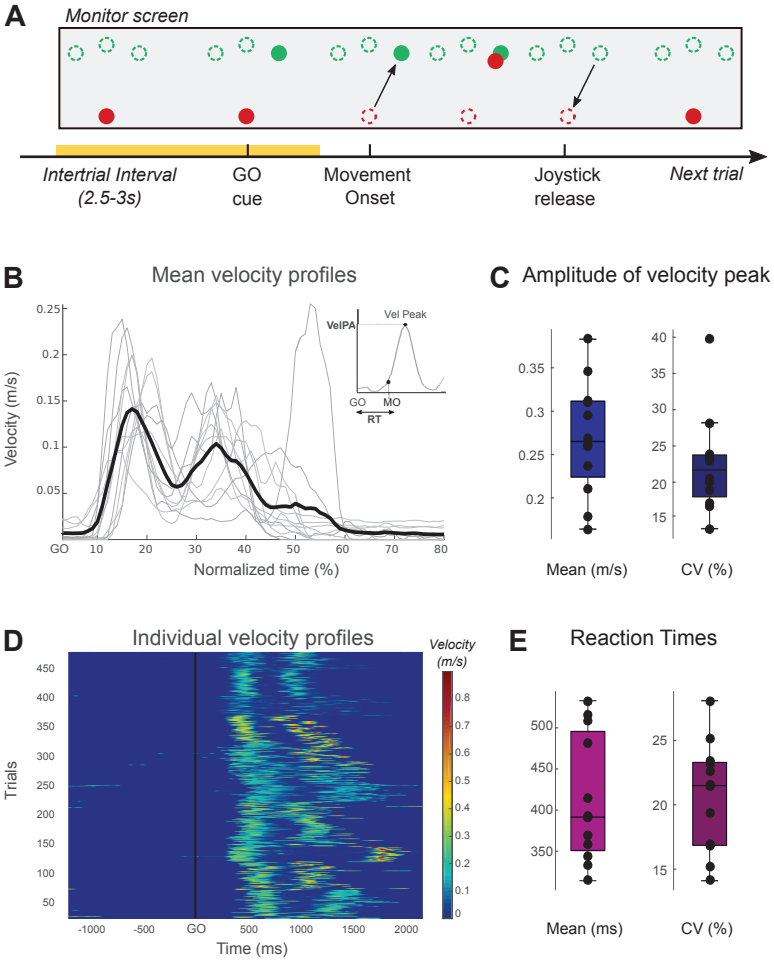
Figure 5: Bursts affect the velocity peak when they are in a critical peri-GO window, with a maximal effect when realigned to Movement Onset. A. Estimated effects and 95% confidence intervals derived from the linear mixed-effects model testing the impact of bursts in 50ms bins on peak velocity. Bins are defined relative to the GO cue, which is indicated by the bold vertical line. **B.** Estimated effects and 95% confidence intervals derived from the same linear mixed-effects model when bins were defined relative to the Movement Onset. Pair of bold vertical lines marks range in which the GO cue would have fallen. Note that for the modelling the velocity peaks are power transformed (see Methods). * Significant model ($p<0.05$) when bins are considered in isolation. Blue shading; significant bins after FDR correction. **C-D.** The majority of the beta bursts occurring in the significant window aligned to movement onset (blue shading Fig 5B) end before the GO cue or right after (yet still have more than half of their duration before the GO). The % of these across subjects are shown ('Before GO') in the panel C whereas the panel D shows the timing of the burst termination points for each subject. *** = $p<0.001$

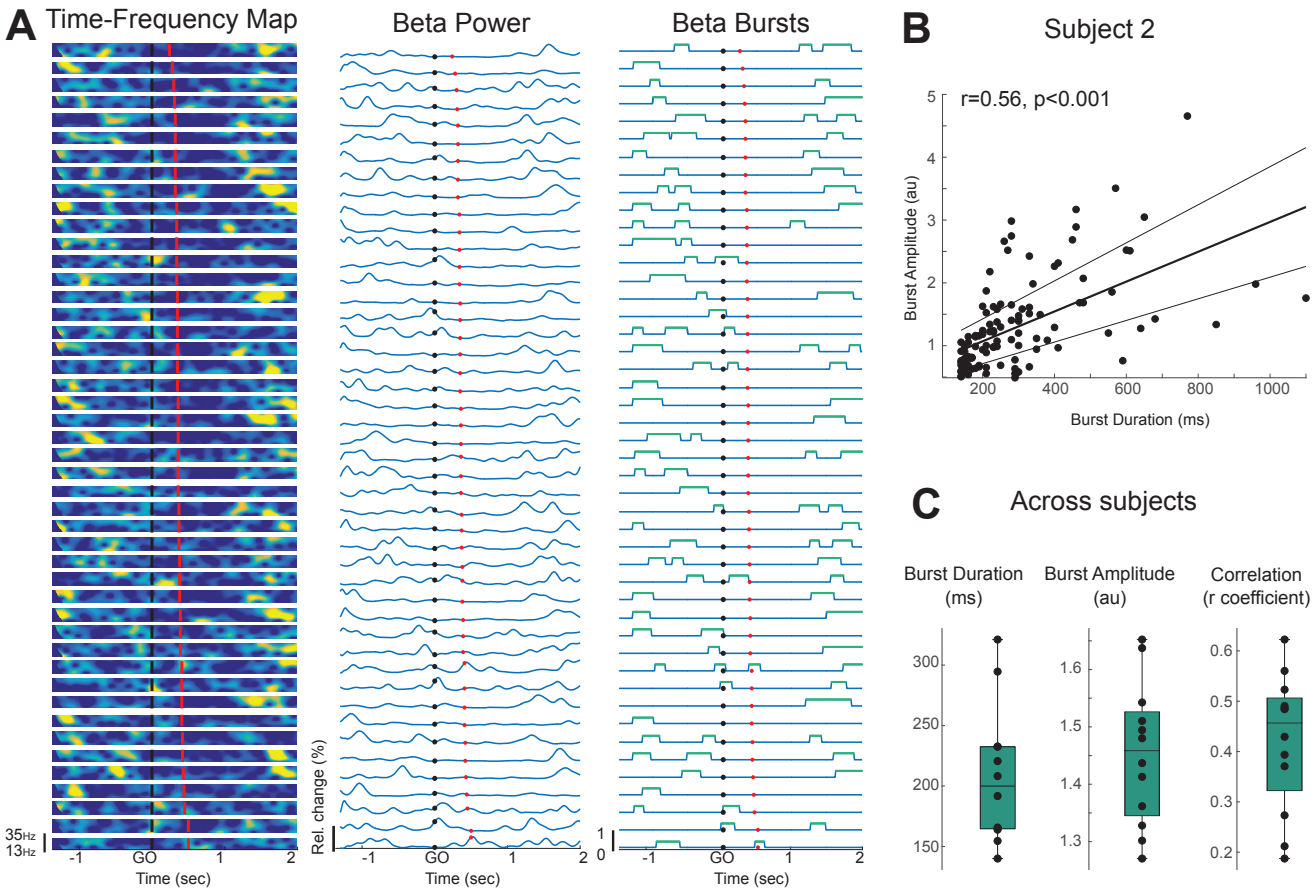
Figure 6: Bursts after the GO cue increase the reaction time, with a maximal effect when realigned to GO. A. Estimated effects and 95% confidence intervals derived from the linear mixed-effects model testing the impact of bursts in 50ms bins on reaction time. Bins were defined relative to the GO cue, which is indicated by the bold vertical line. **B.** Mean

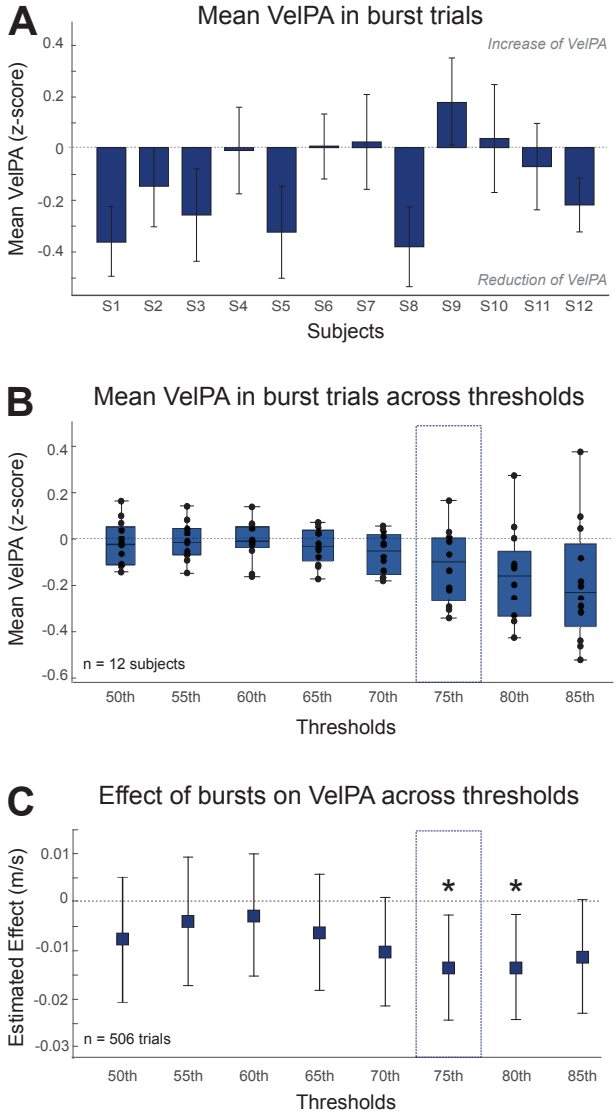
reaction times in burst trials normalized (z-score) to the mean reaction time of all trials for all subjects. A positive value indicates an increase in reaction time in burst trials. Trials are divided according to the presence of a burst in the 200ms post-GO. **C.** Estimated effects and 95% confidence intervals derived from the linear mixed-effects model when bins were defined relative to the Movement Onset. Pair of bold vertical lines marks the range in which the GO cue would have fallen. Note that for the modelling the reaction times were log transformed. * Significant model ($p<0.05$) when bins are considered in isolation. Purple shading; significant bins after FDR correction.

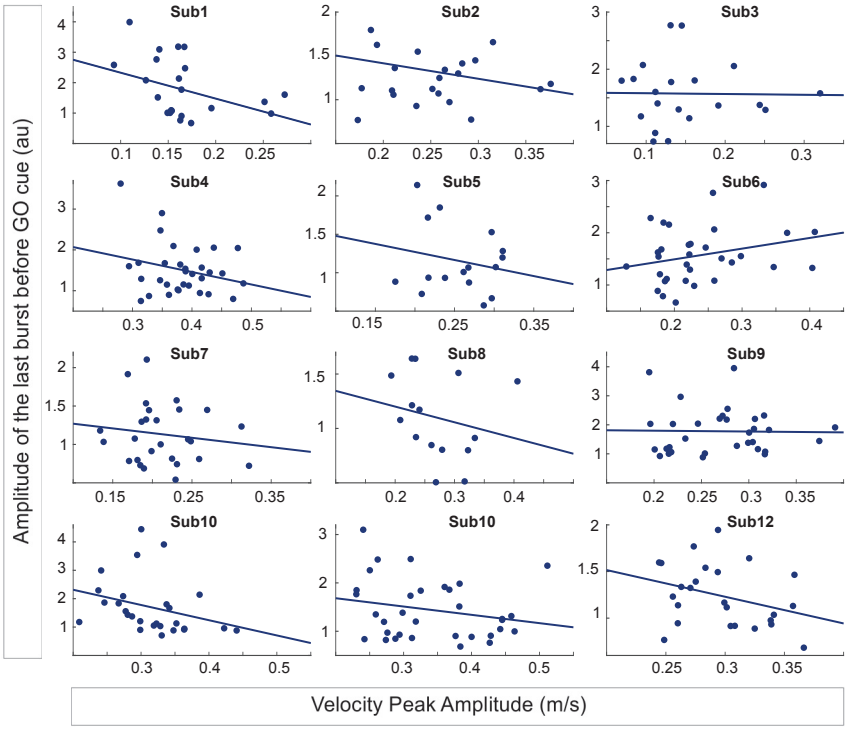
Table 1: Patients details. UPDRS (III), Part III motor score of the Unified Parkinson's Disease Rating Scale. All patients had bilateral implantations. *In Sub4, no signal was recorded for 2 contacts of the right electrode (R3/R4). NA: missing data.

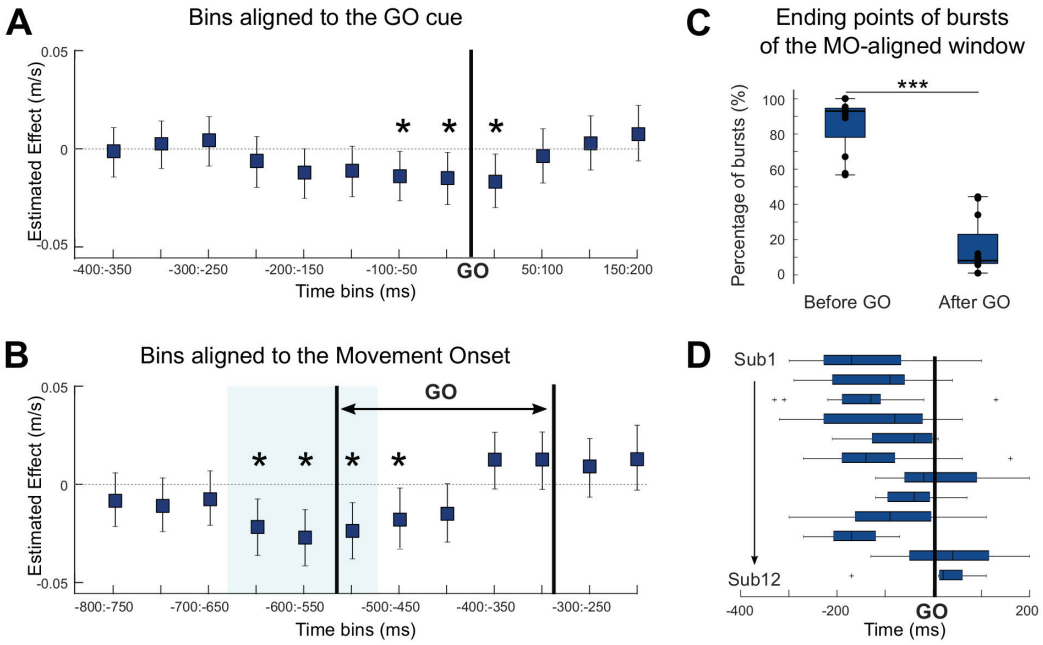
Table 2: Summary of linear mixed-effects modelling results for peak velocity and reaction time. The presence and parameters of beta bursts in the 600ms time window before the GO cue was used as predictors for the modelling. Bursts were included in the model if more than half of their duration was in the 600ms time window. When more than one burst was found in the time window, the amplitude, duration and timing were extracted from the last burst (the burst closest to the GO). If not mentioned, models included all the trials (506 trials). AIC: Akaike's Information Criterion; * significant model after FDR correction ($p<0.05$).

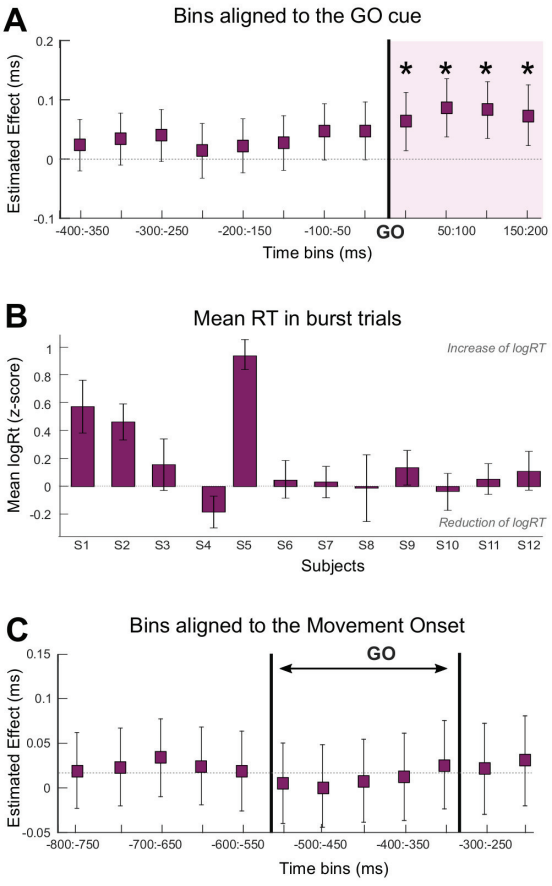












Case	Gender Age(years)	Disease Duration (years)	UPDRS III (OFF)	UPDRS III (ON)	Predominant symptom(s)	Medication (daily doses)
Sub01	F,65	5	33	11	Bradykinesia, tremor	Levodopa, 300mg Amantadine, 200mg Rasagiline, 1mg
Sub02	F,68	14	28	15	Bradykinesia, rigidity	Levodopa, 200mg Ropinirole, 18mg Rasagiline 1mg
Sub03	M,68	13	42	24	Bradykinesia, rigidity , freezing	Levodopa, 500mg Amantadine,100mg Ropinirole, 24mg
Sub04*	M,59	7	61	9	Bradykinesia, rigidity, freezing	Levodopa, 600-1100mg Ropinirole,12mg
Sub05	F,59	14	61	27	Dyskinesia, prolonged OFF periods	Levodopa, 750mg Selegiline,1.25mg
Sub06	M,59	8	49	25	Dyskinesia, freezing, prolonged OFF periods	Levodopa, 850mg Amantadine,100mg Entacapone,1000mg Ropinirole,10mg Rasagiline,1mg
Sub07	M,62	11	63	38	Tremor, bradykinesia, rigidity	Levodopa, 500mg Ropinirole 24mg
Sub08	M,69	9	53	26	Rigidity, bradykinesia	Levodopa, 375mg Entacapone, 800mg Ropinirole, 2mg
Sub09	F,66	17	25	14	Freezing, falls	Levodopa, 375mg Entacapone, 1000mg Amantadine, 200mg Ropinirole, 16mg
Sub10	M,70	11	NA	NA	Tremor	Levodopa, 600mg Entacapone, 1000mg Rotigotine, 4mg
Sub11	F,56	9	49	29	Dystonia, bradykinesia, rigidity	Levodopa, 50mg Apomorphine, 5mg/h Rasagiline, 1mg
Sub12	M,65	6	NA	NA	Tremor	Levodopa, 650mg Rasagiline, 1mg Ropinirole, 21mg

Table 1

Dependant Variable	Predictors	Estimated Effects	t values	p values	AIC	R^2
Peak Velocity	Burst Presence	-1.35E-02	-2.41	**0.0163** *	-1363.4	0.56
	Burst Amplitude	-1.00E-02	-3.19	**0.0015** *	-1367.7	0.57
power transformed	Burst Duration	-5.00E-05	-2.07	**0.0394**	-1361.8	/
	Burst Timing	-3.12E-05	-2.76	**0.0061** *	-1365.1	0.56
	Mean Beta Power	-1.28E-02	-2.16	**0.0313**	-1362.2	0.56
	Burst Amplitude (only burst trials)	-1.32E-02	-2.49	**0.0135** *	-804.2	0.60
Reaction Time	Burst Presence	2.07E-02	1.03	0.3054	-72.7	/
	Burst Amplitude	1.75E-02	1.55	0.1128	-74.1	/
log transformed	Burst Duration	9.00E-05	0.99	0.3204	-72.6	/
	Burst Timing	9.80E-05	2.34	**0.0168** *	-77.4	0.42

Table 2